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CORRESPONDENCE

Tumor lysis syndrome after transarterial chemoembolization plus portal venous embolization for hepatocellular carcinoma

Wan-Ling Tsai [a], Po-Chin Liang [b], Chien-Hung Chen [a,*]

[a] *Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan*
[b] *Department of Medical Imaging, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan*

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Tumor lysis syndrome occurs mostly in patients with hematologic disease.[1,2] Solid tumors are rarely affected by tumor lysis syndrome because they are relatively resistant to cytotoxic therapies. Mortality rate in patients with solid tumors is higher because of delayed diagnosis.[2] A 51-year-old man with chronic hepatitis B was admitted for large hepatocellular carcinoma (HCC). Abdominal computed tomography showed liver cirrhosis and a 17-cm hepatic tumor at S7/S8 with contrast enhancement in the arterial phase and washout in the venous phase. No evidence of portal vein thrombosis was noted. Abdominal magnetic resonance imaging showed similar results. Preoperative portal venous embolization (PVE)[3] and transarterial chemoembolization (TACE) were used to decrease the tumor burden. Two days after TACE, elevation of aminotransferase, bilirubin, uric acid, creatinine, and lactic dehydrogenase levels were noted (Table 1). Tumor lysis syndrome with acute kidney injury was diagnosed.[2,4] Renal and liver function gradually improved after adequate hydration and rasburicase administration. Left lobe hypertrophy after PVE was observed; however, the volume of left lobe was not enough for major hepatic resection. Two further sessions of TACE were performed for the residual tumor without recurrence of tumor lysis syndrome. The patient was followed up at an outpatient clinic. The common risk factors for tumor lysis syndrome are tumor burden, cancer lysis potential, baseline renal insufficiency, dehydration, hyperuricemia, hypotension, and exposure to nephrotoxins. In previous case reports about TACE-induced tumor lysis syndrome,[5] a large tumor burden (>5 cm) is the common feature. In this case, PVE and large tumor burden led to increased tumor necrosis and subsequent tumor lysis syndrome. When tumor lysis syndrome develops, treatment with aggressive hydration, allopurinol, rasburicase, and urine alkalinization is warranted.[1,2] Aggressive hydration can rapidly improve renal perfusion and glomerular filtration and minimize acidosis. Loop diuretics can be used to maintain adequate urine output. Rasburicase is more effective than allopurinol in preventing xanthine accumulation and directly breaking down uric acid. Urine alkalinization can be considered when rasburicase is not available, but it must be discontinued when hyperphosphatasemia develops. In addition, electrolyte, creatinine, and uric acid levels should be monitored frequently (every 4–6 hours in high-risk groups). Tumor lysis syndrome is not normally considered among the complications of TACE because of its low incidence. Thus, mortality rates are high because of delayed diagnosis. With early detection and treatment, our

* Corresponding author. Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, 7, Chung-Shan South Road, Taipei 100, Taiwan.
E-mail address: chenhcc@ntu.edu.tw (C.-H. Chen).

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Table 1 Serial laboratory data before and after TACE.

	Reference range	Baseline	After PVE	After TACE						
			3 d	2 d	4 d	9 d	12 d	15 d	42 d	80 d
AST	<37 U/L	71	73	9349	486		52		64	46
ALT	<41 U/L	60	61	691	169		46		50	27
T-bil	0.2—1.2 mg/dL	1.1	1.3	7.1	7.8	8.4	4.5	3.8	1.5	1.0
BUN	4.5—24 mg/dL	21		42	73	135	123	79	20	20
Cr	0.6—1.3 mg/dL	1.3		4.1	7.5	5.4	3.6	2.6	1.6	1.5
Na	135—148 mmol/L	135		121	126	133	133			133
K	3.5—5.3 mmol/L	4.2			4.4	3.9	4.8			4.3
Ca	2.02—2.6 mmol/L				1.86	2.28				2.3
P	2.7—4.5 mg/dL				7.2	7.6		3.1		3.2
UA	3.5—7.5 mg/dL			15.5			9.2			
LDH	230—460 U/L			33820			605			362

ALT = alanine aminotransferase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; Ca = calcium; Cr = creatinine; K = potassium; LDH = lactic dehydrogenase; Na = sodium; P = phosphate; PVE = portal venous embolization; T-bil = total bilirubin; TACE = transarterial chemoembolization; UA = uric acid.

patient recovered without incident. Therefore, tumor lysis syndrome must be considered in patients undergoing TACE for HCC. Early detection and intervention can reduce mortality rates because of tumor lysis syndrome.

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